Thursday, March 22, 1990
8:30AM–10:00AM, Room 43
Valvular Heart Disease: Natural History and Complications of Valve Replacement

Optimal Timing for Surgical Correction of Chronic Mitral Regurgitation to Preserve Left Ventricular Performance.

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The ability to predict the long-term response of left ventricular (LV) size and performance to mitral valve replacement (MVR) in patient (pt) with chronic mitral regurgitation (MR) has remained elusive. To test the hypothesis that when integrated LV chamber performance is abnormal but myocardial properties are normal would identify these pts, who might benefit in this regard from earlier MVR, we studied 32 control pts and 18 pts with MR using micromanometer LV pressures and radionuclide angiograms to calculate LV chamber elastance (Emax) and biplane contrast cineangiograms to calculate myocardial circumferential stress-shortening (σ_c-EF) relations. The MR pts were then divided into 3 groups (G): GI-normal Emax and σ_c-EF, GII-reduced Emax but normal σ_c-EF, and GIII-reduced Emax and σ_c-EF. Following MVR, MR pts were evaluated with radionuclide LV EF determinations at 3, 6, and 12 mos. These data are:

	Pre-op	Post-op		
		3mos	6mos	12mos
Group I (n=6)	62±9	65±6	62±9	63±9
Group II (n=8)	62±6	51±8+	53±8+	57±5
Group III (n=4)	40±9**	29	29±1	29

**p=0.001 vs GI and II; +p<0.05 vs pre-op and GI. These data suggest that in pts with chronic MR when the pre-operative LV chamber elastance is reduced but myocardial properties are preserved may be the optimal time for MVR to preserve LV EF at 12 months, despite an early, significant reduction in LV EF at 3 and 6 months.

ASSESSMENT OF MITRAL VALVE ORIFICE AREA AT REST AND FOLLOWING CHANGES IN LOADING CONDITIONS IN HUMANS.

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The Doppler-echocardiographic transmitral velocity profile has been used to assess LV filling employing the following relationship: flow(cm^3) = time velocity integral (TVI) [cm] x cross-sectional area (CSA) [cm^2] of the mitral valve. This method for measuring transmitral flow assumes that the mitral valve CSA remains constant during diastole. However this hypothesis has never been directly tested in humans. If transmitral flow can be independently measured, then MVA can be calculated as: MVA (cm^2) = flow (cm^3) / TVI (cm). In order to assess LV filling directly on a beat-to-beat basis, we used an impedance catheter which has previously been shown to accurately measure changing LV volume. The impedance catheter and Doppler transmitral velocity waveforms were recorded simultaneously. Volume changes and the TVI were calculated at 80 or 120 ms intervals in three cardiac cycles. We studied 14 subjects with normal mitral valves and no aortic insufficiency. In 7 subjects the study was repeated after a sublingual nitroglycerin.

Results: MVA, as calculated by this method, varied significantly during diastole. The maximal MVA was 5.8+/- 0.2 cm^2 while the minimal MVA was 2.4+/- 0.2 cm^2 ($p<.001$). In all cases MVA was greatest in early diastole, becoming smaller in mid-diastole, and larger again in late diastole. Sublingual nitroglycerin produced a clear decrease in early LV filling rate, early transmitral velocity, LV end-diastolic volume, and in LVEDP (p all <.05). However, this was not associated with a change in maximal MVA, minimum MVA, mean diastolic MVA or the MVA during the first 240 ms of diastole (p all>0.5).

Conclusion: These data demonstrate that MVA is not constant during diastole. The data further suggest that MVA is neither preload nor filling rate dependent. This raises the possibility that mitral valve motion may not be dependent upon transvalvar gradient or flow. The demonstration that MVA is not constant during diastole also has implications for the the use of Doppler-echocardiography in the calculation of transmitral flow, as changes in TMV may occur secondary to changes in MVA rather than changes in flow.

NATURAL HISTORY OF ASYMPTOMATIC SEVERE MITRAL REGURGITATION: DISSOCIATION OF SYMPTOM DEVELOPMENT AND VENTRICULAR PERFORMANCE?

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The rate of progression to surgery-requiring disease among pts with hemodynamically severe mitral regurgitation(MR) is poorly defined. Knowledge of these data would help to determine optimal followup schedules. Therefore, we followed 39 pts with echocardiographically severe MR, minimal or absent symptoms, and normal LV and RV performance (i.e., radionuclide cineangiographic LV ejection fraction[EF]≥45% and RVEF≥35%) with annual clinical and radionuclide examinations for 2-8 yrs to determine the rate of progression to intolerable symptoms (NYHA Class≥III) and/or EF levels previously shown by us to indicate high imminent mortality risk (LVEF≤45%, RVEF≤30%). During an average 3 yrs of followup, 12 of the 39 pts reached criteria for operation: 1/12 developed low EF without symptoms and 11/12 developed NYHA Class III symptoms; one of the latter had RVEF=17% at symptomatic presentation, following a 3 yr hiatus in objective testing. Cutter-Ederer life table analysis indicates that, after 7 yrs followup, probability of deterioration to operable state is 50%. These findings indicate moderately rapid progression to operability among asymptomatic pts with severe MR, suggesting the need for periodic clinical evaluation. In addition, however, objective deterioration to high risk state can develop before or without symptomatic deterioration. Therefore, periodic objective testing must supplement clinical evaluation in MR.

INCREASED INCIDENCE OF PERIOPERATIVE MYOCARDIAL INFARCTION IN VALVE REPAIR FOR MITRAL REGURGITATION.

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Mitral valve repair (REPR) is preferred to prosthetic replacement (MVR) based in part on preservation of LV function. We studied the incidence of myocardial infarction (MI), a determinant of LV function, in 335 consecutive pts with normal coronary arteries having REPR (n=224) or MVR (n=111). MI was based on diagnostic ST-segment elevation of≥ 1.5mm (ECG+), and CPK≥ 1000, CPK-mb≥ 133, or SGOT≥ 100 IU/L. Follow up ECGs were obtained to determine if new Q-waves (QW), T-inversion (TI), or resolution of the acute ECG had developed. 2-D echocardiograms for segmental wall motion abnormalities (SEG) were obtained in all MI patients.

RESULTS	ECG+	QW+	TI+	R+	CPK+	MB+	SGOT+	SEG+
REPR	12	7	2	3	12	11	9	3
MVR	2	0	0	0	0	0	0	1

Thus 12/224 (5.4%) REPR had ECG and enzyme evidence of MI, while 0/111 (0%) MVR had similar findings. (p=0.01). All 12 MIs involved the inferior ECG leads, and all SEG were in the inferior wall. We hypothesize the etiology to be coronary air emboli into the right coronary artery introduced during LV insufflation during testing of MV leaflet coaptation in the presence of antegrade cardioplegia with the LA open. Retrograde cardioplegia and better aortic/LV venting may lower this complication rate. No pt developed hemodynamic compromise, nor was their post-op recovery prolonged due to this event, indicating a small amount of residual damage. We remain enthusiastic about the advantages of valve repair for mitral regurgitation.